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MEDICINE AND THE COMMUNITY*

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It is with very sincere feelings that I wish to express my appreciation of the honour of being elected President of the Medical Association of South Africa, and my thanks to my colleagues in the Northern Transvaal Branch and the Federal Council and the Association for the distinction which you have conferred on me. When I think of some of my predecessors in this high office, who have given signal service to the Association and to the profession, I feel that I must strive to maintain that high tradition.

Ek sal ook graag my dank wil betuig aan daardie mediese kollegas in Pretoria wat gehelp het om hierdie Kongres te organiseer, en ek is seker dat hulle van hierdie Kongres 'n baie groot sukses sal maak. Die Organiserende Komitee was uitstekend en dit was 'n plesier om saam met hulle te werk. Die dames-komitee was baie bekwaam en betroubaar. Vir almal wat gehelp en gewerk het, wil ek namens die Mediese Vereniging baie, baie hartlik bedank.

Verder wil ek ook my dank oorbring aan die Burgemeester en die Stadsraad van Pretoria, die Stadsklerk en ander beamptes, vir hulle hulp en bereidwilligheid.

Ook ons hartlike dank aan die Rektor van die Universiteit en sy personeel vir die gebruik van die pragtige geboue en Universiteitsgronde wat hulle tot beskikking gestel het vir ons kongres-vergaderplek en wetenskaplike vergaderings. Graag wil ek ook my besondere dank uitspreek aan Sy Edele die Minister van Gesondheid vir die eer wat hy ons bewys het om ons Kongres te open.

In a medical congress like this, when we develop our plenary sessions and scientific meetings, and also fill a business and social order, I think there is, behind these activities, a deeper desire to give expression to our professional strength and solidarity and, for a time, to see and experience the profession as a whole.

Dr. J. H. Struthers

MEDICINE A CENTURY AGO

As we are celebrating the centenary of Pretoria, I thought I would invite you to consider with me something of the conditions which prevailed in the medical profession, and something of the state of medical knowledge and practice a hundred years ago, contrast this with the position we find ourselves in today, and consider some of the implications this may have for the community.

It is 99 years since our

* Presidential Address delivered at the adjourned Annual General Meeting of the Association, Pretoria, 17 October 1955.

parent association, the British Medical Association, was founded in 1856—prior to which, for 24 years, it had existed as the Provincial Medical and Surgical Association.

Its founder was Dr. Charles Hastings of Worcester (England) and it would be instructive to take note of his medical education. After a period of apprenticeship, and having attended a private school of anatomy, he became, at the age of 18 years and with no medical qualification, a house surgeon at the Worcester Infirmary. He was elected to this post by 134 votes to 133. Some years later he graduated in medicine at Edinburgh. At this time there was no hard and fast line demarking the qualified from the unqualified doctor. In England in 1846, when the first medical directory was published, the number of unqualified doctors was twice the number of qualified, and this was the case until the Medical Act of 1858 produced order and a Medical Council and a Medical Register.

Surgery. In the first half of the 19th century, surgery could scarcely be considered a fine art. There were no anaesthetics, the patient was bound to the operating table whilst the surgeon worked with septic instruments and unwashed hands. However, although methods were primitive, there was room for wide variations in skill. Speed was really almost the essence of success. When Mr. Brandsby Cooper of Guy's Hospital took 55 minutes to perform an operation for the removal of stone and, despite being bled by leeches, the patient died the next day, the *Lancet* published a gruesome account of the operation and accused Mr. Cooper of being an incompetent surgeon and losing his nerve. Mr. Cooper sued the editor of the *Lancet* for libel which resulted in a surgical *cause célèbre*. Learned counsel demanded £2,000 damages—a goodly sum in 1829. The owner and editor of the *Lancet*, Mr. Thos Wakley, defended himself and, in doing this, he quoted John Hunter as saying that bad surgeons were like bad carpenters—they made work for one another—and the good surgeon would starve if it were not for the unskilful ones. There was a long and strained waiting for the jury's verdict, which was ultimately for Mr. Cooper, with damages of £100.

At this time we note that the Cape Western Medical Association decided to discontinue its subscription to 'the publication entitled the *Lancet*, owing to its offensive scurrility and personality.'

A hundred years ago surgery was still very much in the state in which it had existed right from the middle ages, but it was on the brink of mighty transformation. The introduction of anaesthesia—a name coined by Oliver Wendell Holmes—was at hand. Ether was first used as an anaesthetic by Dr. Morton in America in 1846, and Dr. Simpson introduced chloroform in Scotland in 1853. This conquest of pain completely altered the field of surgery. Within 20 years the work of Pasteur inspired Lord Lister to introduce his antiseptic methods and thus modern surgery, as distinct from medieval surgery, was launched. For the past 50 years, radiology has been surgery's handmaiden, and in the past 20 years, infection control with antibiotics and the enormous strides made by modern anaesthesia have again revolutionised modern surgery. One may say that many of the routine surgical procedures of today were hardly dreamed of even 25 years ago.

Nursing. It is also just 100 years ago, in 1855, that Florence Nightingale went out to the Crimean war. Her powers of observation and insight, and her intelligence and determination, were essential to the launching of the nursing profession—the existence and development of which has been essential to the development of present-day medicine and surgery.

Medicine. Graves introduced the timing of the pulse with a watch in 1846, and it was only following this that the ophthalmoscope, the laryngoscope and the clinical thermometer came into use.

In the department of medicine a hundred years ago, very little was understood of the disease process. The chief method of advancing medical knowledge was a careful study of conditions which could be observed and characterized in the patient during life, and the regular association of this with changes found in the different organs after death. Thomas Addison of Guy's was the great exponent of this clinico-pathological method, which resulted in his publication in 1855 of his classical description of Addison's disease, and also of Addison's or pernicious anaemia.

Following this, came the scientific method of observation and experiment, an excellent and early example of which was the work of George Murray, my Professor of Medicine and medical chief. He was the first to use extract of thyroid gland for the treatment of myxoedema. It was in 1891 that he arranged with a slaughter-house to be allowed to dissect out sheeps' thyroid glands. The Newcastle Medical School refused him a room for his experimental work because they disapproved of it, and Armstrong College came to the rescue. He injected regularly an extract of thyroid into a myxoedematous woman 46 years old and demonstrated the patient, now active and sprightly, to the Newcastle Medical Society. The patient lived to the age of 74, by which time she had received the equivalent of 870 sheeps' thyroids.

It was only during the present century that the conception of chemical regulation and co-ordination of bodily functions has found a place. The idea that health, and even life itself, depends on the liberation of minute amounts of chemical substances into the blood stream, ultimately gave us cortisone and vitamin B12, which have now provided the therapeutic answer to the conditions described by Addison 100 years ago.

Especially with the assistance of the physiologist and bio-chemist, new fields have opened up, new paths of experimentation have been followed, and we have today, for our clinical use, a wide range of most effective and potent drugs.

In 1855 there was no knowledge of the nature of infectious diseases. Typhoid fever was still epidemic. Cholera swept Europe in epidemics every 10 years or so from an endemic centre in Asia. The Prince Consort died in 1861 of typhoid fever at the age of 42 years. In that year, John Simon was appointed as the first Medical Officer of Health, to the City of London, which post he held part-time, together with the position of Surgeon to St. Thomas Hospital. The reports which he promptly began to issue on the sanitary scandals of the City, stirred the public conscience about matters of simple

common decency in hygiene. Let me quote: 'This is evil enough, but worse remains behind. It is no uncommon thing in a room 12 feet square or less, to find three or four families styed together, filling the same space night and day—men, women and children—in the promiscuous intimacy of cattle'. And again: 'Notwithstanding all that Medicine can achieve to succour the body as it struggles against actual disease—sanitary law embodying the principles of preventative medicine may ensure to the aggregate masses of the community prolongation of life and diminution of suffering—in the working of some single enactment it may affect the lives of generations of men and may moderate, in respect of millions, the sources of orphanages and poverty'.

Following such advocacy and the discoveries of medical science, the progress in matters of public health in the past 100 years has been phenomenal. Most infectious diseases have been conquered, if not abolished. We may be about to see poliomyelitis brought into this category of conquered diseases. The World Health Organization began a new era of international co-operation in 1946. Already it has made frontal attacks on malaria, yaws and venereal diseases in Asia and, by supplying the drugs and equipment and instructing the native inhabitants in their use, it has achieved great success. The late Director-General of WHO, Dr. Brock Chisholm, had this message handed to each new member of his secretariat: 'We must think and act in terms of mankind as a whole. We must be ready to give up old ideas, certainties and devotions in order to place the welfare of all people everywhere on the same level of values, regardless of where on this little earth one happens to have been born himself. In other words, we must try to attain an equal degree of loyalty to all members of the world community, irrespective of race, religion and colour and any other group characteristics'.

Until 100 years ago, the struggle for survival was the same in the human species as in any other. Death was the major selective force. Rarely did the majority of babies born live to become parents—as many as half the babies might die before their first birthday. The pattern of reproduction in the world was cruel and wasteful, but it was the basis of maintaining generic fitness. Now a great change has occurred.

END-RESULTS OF MEDICAL SCIENCE

Medical progress began to provide the tools which controlled the human life-process. What with sanitation, public-health measures, modern surgical and obstetrical techniques, vaccines, inoculations, antibiotics, we now have a new era which could be called the Vital Age. Now we are in a position to control life and death to a considerable degree. Bertrand Russell put it very tersely when he said: 'Broadly speaking, we are in the middle of a race between human skill as to means and human folly as to ends. Given sufficient folly as to ends, every increase in the skill required to achieve this is to the bad. The human race has survived hitherto owing to ignorance and incompetence; but given knowledge and competence combined with folly, there can be no certainty of survival. It follows that, unless men increase in wisdom as much as in knowledge, increase in knowledge will be increase in sorrow'.

There is a story of two well-meaning members of the staff of the Institute of Tropical Agriculture in Trinidad who went on a war-time visit one week-end to friends in Georgetown, British Guiana. Whilst there, they heard from the Resident Health Officer of the awful infantile mortality rate amongst the native population—350 per 1,000 in the previous year. The most important cause, they were told, was insect-borne diseases. They were horrified; and then they made the suggestion that DDT should be used, and explained how, by dusting it from planes, almost complete insect control would be possible. The Health Officer took this up and, between 1945 and 1948, an area 10 miles square, including Georgetown, was periodically sprayed with DDT. Results were almost instantaneous, and today the net rate of increase in population is such that the population will double itself in 10 years. The economy of British Guiana affords the native population little more food than 1,500 calories a day, which is the point of famine. Unless a new economy can be established or ever-increasing shiploads of food sent there, the DDT suggestion may become a very grim thing for the natives.

Over the entire world 68,000 people are added to the population daily; in other words, the increase in world population is now 25 millions per year. During the 19th century, the century of medical awakening, the world population doubled. The sparsely populated lands of the west had already been discovered; now they were populated. By 1900, world population was given as 1,500 millions. In the present century the increase has continued apace—the present mid-century population of the world is computed to be 2,500 million and at the present rate of increase it will be 4,000 million by the end of the century.

POPULATION PATTERNS

The changing patterns of birth and death which have emerged in the western world in the past century have been mainly brought about as a result of the progress of medical science. (They affect not only the numbers of people, but also the kind of people, their age, sex, ancestry and cultural background.)

On the basis of birth rates and death rates, Dr. Warren S. Thompson, Director of the Scripps Institute of Population Research, groups the countries of the world in 3 categories:

Class 1 populations include most of Europe and North America. They represent one-fifth of the world population. Expectation of life due to the low death rate is nearly 70 years. The standard of living is high, the birth rate low.

Class 2 are the countries in the process of transition—Latin America, the Balkans, Russia and Japan. They represent one-third of the world population. Public-health measures have started a decline in the death rate, but the birth rate remains high. Soviet Russia is the one country in the world actively promoting a larger population.

Class 3 populations, which are typified by China and India, include most of Asia. They represent more than half the world's population. Their expectancy of life is 30 years.

In Western Europe, the average number of births per woman just exceeds two. In China, the average is over 7; and in China, as the expectancy of life is 30 years, this greatly increases the number of people in the population at the height of their productive powers.

Following on this, there are 3 very important problems now confronting this community and the communities of the world. The *first* is to increase and improve agriculture. Marquis Mirabeau, the French revolutionary statesman, put the matter concisely as follows: 'The State is like a tree—the roots are agriculture. The trunk is population. The branches are industry. The leaves are commerce and art. It is to the roots that a remedy must be applied if the tree is not to perish'.

Of the world's people 70% live by agriculture. Their production must increase. Soil erosion must be controlled. Soil fertility must be increased. Great strides can be made to control plant diseases, insect and rodent infestations and spoilage and to improve transport and marketing.

The *second* problem that must be faced is that of human fertility. Here the old taboos are beginning to weaken. But the question is not easy. In the low-birth-rate countries, i.e. the Class I countries, the spread of birth control amongst the more privileged has established existing birth differentials which are already a serious threat to future genetic quality. The future increase of birth control in this class might well be disastrous. On the other hand, excess fertility throughout the world *could* be controlled. It is a question of giving the people—the ordinary people—the facts and allowing them to make their decision. The Japanese people are desperately trying to do it today, despite the opposition of political and other leaders. The Africans, the Chinese, the Indians, can also do it. The community must see that education and the knowledge is universal, and then in matters of birth control, having the knowledge, the people must decide.

The *third* is the problem of the ageing community. The effect of our being able to increase the average span of life during the 20th century by as much as 20 years, is that the number of people over 65 years has increased twice as fast as the population as a whole. In England the proportion of people over 65 to those of working years is given as 1 to 7. In 25 years' time the proportion is expected to be 1 to 3½.

THE AGEING PROCESS

In considering various aspects of the ageing process, one or two things should be considered.

Firstly, the considerable gap between the official and the natural onset of old age. The official onset is 60 for men and 55 for women, although this has been increased by 3 years in some quarters. Dr. John Sheldon, in his Wolverhampton survey, considered that natural old age makes itself felt by about 70 and by 75 it has acquired such momentum that few escape.

Secondly, women outnumber men in advanced ages, their average longevity being longer by 3 or 4 years. Further, the two sexes receive medical treatment in equal degree between 60 and 70, but thereafter men's requirements decrease and women's increase so that, by 85 years, women have double the medical attention of men. Hence we may say that, although in old age men tend to maintain a better physique than women, their hold on life is brittle whilst, with women, despite increasing decrepitude with the years they have a power of sheer survival denied to men.

In a Wolverhampton survey of the aged in 1947, the following facts emerged: The number of old people in general and mental hospitals and institutions was less than 3% of all old people. However, of the 97% living in rooms and private houses, 7% were imposing strains of the severest character on those who looked after them, robbing them of a chance of normal life. Further, 10% of old people were confined to home on account of physical or mental decrepitude. These facts suggest that the problem of the aged will increase in importance, as will the responsibility of the community. What can be done might be considered in the light of the following main objectives:

1. We must endeavour to preserve the independence of old people as long as possible so as to stave off the day when they become a burden to others. To do this, ways should be considered of enabling the man to work until the natural onset of old age, so that he may be retained as a useful citizen as long as possible.

2. The principle that the family should be responsible for old-age care is undoubtedly right, and creates greater happiness and security for the aged. However, adequate accommodation should be available when they become sick and require to be hospitalized. District nurses, if made available, could by their nursing assistance, often make hospitalization unnecessary. When necessary, provision should be available for them in local general hospitals, so that they can be visited by their relatives and friends, but unless special accommodation is set aside they are greatly handicapped in getting accommodation in competition with acute cases. On the other hand, wards for the chronic sick should be cheaper to run and staff than acute wards.

3. The community must do something to assist the family in the care of aged dependants. The Red Cross, St. John's and Noodhulpliga would do wonderful work if they could organize home aids and auxilliaries to assist with this increasingly important work. Help to the aged certainly pays dividends in human comfort and content. In the field of gerontology and geriatrics there is a great field for medical research in South Africa.

SPECIALIST AND FAMILY DOCTOR

So far I have considered the effect of modern medicine on the community. Now, before I close, I should like to consider some of the problems it raises for the individual.

With the widening of the basis of medical scientific knowledge in every field, the specialist is becoming more and more a necessity. Not only that, but general consultants, particularly in medicine and surgery, look as though they will also be gradually replaced by the specialist consultant who deals with a small aspect of medicine or surgery. The acquiring of knowledge and special skills in limited fields brings with it great technical efficiency and encourages research, and this is the trend of present-day development.

However, I believe that the part played by the general practitioner in bringing the fruits of medical knowledge to the community should not diminish, but should increase.

The most responsible and difficult diagnosis may very well still be that between the early symptoms of organic disease and those of functional origin; whether to investigate or to treat symptomatically may be a decision entailing life or death. To seek the aid of the growing power of medicine in order to prolong life might merely add to the suffering to be endured. The problem which confronts the patient, and also his doctor, may be scientific or psychological or moral, or all three. For instance, the different psychological types are liable to develop different variations of physical illness, the aggressive type being liable to develop cardiovascular troubles and the retrogressive type digestive disturbances. Further, illnesses are often purposeful, providing a patient with a way out of a dilemma which cannot be solved in another way. Again, we can now prevent birth or terminate pregnancy, control infection, suppress or increase instinct, produce changes of mood with electricity, modify the reactions of the mind with drugs, and influence the personality with the knife. In facing the problems which may arise, the patient and his relatives are still in need of a family doctor who, seeing his patient and his illness as a whole, can with wisdom advise.

In order satisfactorily to fulfil this role, the family doctor's training should continue throughout his medical life. He is by far the largest group within the profession, yet he is completely excluded from the teaching hospitals and postgraduate facilities and refresher courses available for him are very limited. One real difficulty is that he is overworked, his services are not sufficiently highly valued, and he has little or no leisure. He must always be cheerfully available and his own convenience must be continually sacrificed for those of his patients.

On the other hand, I believe that the role of general medicine will more and more belong to him, and much of his future will depend on his becoming a better doctor. We must remember that prudence governs the employment of science and that, in the last resort, we are concerned with the human person whom science by itself is inadequate to comprehend.

And now I have finished. If I were to try and sum up, remembering some of our great predecessors in the profession, I would say that over the past 100 years our interests have remained the same as theirs—the enlargement of man in length of days, in usefulness and in happiness. It has always been that length of days is the measure.

The passage with which I conclude is enshrined in a report of William Farr, the compiler of vital statistics, who wrote a hundred years ago: 'The prolongation of the life of the people must become an essential part of family, municipal and national policy. And so the mind is involuntarily disposed to listen to the voice of the prophet Isaiah:

'"And the voice of weeping shall be no more heard, nor the voice of crying. There shall be no more thence an infant of days, nor an old man that hath not filled his days; for the child shall die a hundred years old. And they shall build houses and inhabit them; and they shall plant vineyards and eat the fruit of them. They shall not build and another inhabit; they shall not plant and another eat; for as the days of a tree are the days of my people, and mine elect shall long enjoy the work of their hands"'.

DR. J. H. STRUTHERS

Dr. J. H. Struthers, the newly-elected President of the Medical Association of South Africa, was born in England in 1896. His medical course, which he took at Manchester University, was interrupted by 3 years' service in World War I as Surgeon Sub-Lieutenant, R.N.V.R., mostly in destroyers, in 1915-18. He qualified M.B., Ch.B. in 1922.

Dr. Struthers was engaged in general practice in Lancashire during 1925-39, and held the position of Honorary Surgeon, Bury Infirmary during 1926-39, and on retiring was appointed as Consulting Surgeon. He was President of the Bury Division of the British Medical Association in 1938-39.

Since 1939 Dr. Struthers has been in private practice in Pretoria. He was Honorary Assistant Surgeon of Pretoria General Hospital from 1940 to 1945, and had charge of a military ward (part-time) during World War II in 1940-43, with the rank of Major, S.A.M.C.

Dr. Struthers was President of the Medical Association of South Africa in 1949, and has been member of Federal Council since 1947 and of the Executive Committee of Federal Council since 1948.

ABSTRACT : UITTREKSEL

Njovera. Wilcox, R. R. 1955. Central African Journal of Medicine. 1, 1, 30.

The author describes this condition as it occurs among Africans in Southern Rhodesia. He notes the similarity to bejel, and considers that both are examples of endemic syphilis. The lesions are commonly those of secondary syphilis in children, and gummatous lesions in those of later age.

The Natives differentiate njovera from venereal syphilis, which they sometimes refer to as 'the white man's disease'.

F.W.F.P.

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EDITORIAL

THE LIFE SPAN

The duration of life is dependent on genetic and environmental factors. The influence of genetic factors in man was first suggested by the observation that some families are long lived and others short lived. However, special study of animals has shown that there are differences in the life span of closely related animals of different genetic constitution living under the same environmental conditions.

Various species of animals have been used to determine the relationship between growth and development and the duration of life. In their studies investigators have tested the effects on longevity of promoting or retarding of growth through the use of special diets. The role of growth in relation to life span has led to much controversy. Retardation of growth would not seem to promote longevity and accelerated growth does not appear to shorten the life span. The concept that is favoured is that growth and longevity are not interdependent but are closely related to the rate of metabolism in general, and therefore may be coordinated phenomena governed by the same metabolic principles.

Since life is terminated in most cases by disease, the important factors that need consideration in connection with the life span are those concerned with the promotion or prevention of disease, and with the acceleration or retardation of ageing and of growth.

Undernourishment and overfeeding both have a harmful effect on the course of disease, but the relation of diet and growth to life span is more controversial. An immediate difficulty is the question of what constitutes an optimal or normal diet. It is doubtful whether the same diet has the same influence on growth, on breeding and on longevity. Different individuals have different requirements, and there are other obvious difficulties. Food consumption varies with the season, and with the quality of the diet; thus, for example, synthetic or purified diets have been shown experimentally to be inferior to natural foodstuffs. In laboratory studies the strain of animal used, sex differences, the temperature of the laboratory, the question of crowding, or the change of feeding habits by single caging, are all important considerations in life-span experiments. The difficulty is increased when observations extend over many months or years.

With some laboratory animals there is apparently little doubt that moderate dietary restriction during

VAN DIE REDAKSIE

DIE LEWENSDUUR

Die duur van die lewe word beheer deur genetiese en omgewingsfaktore. Die invloed van genetiese faktore op die leeftyd van die mens was eers gesuggereer deur die waarneming dat sommige families 'n lang en andere 'n kort lewensduur het. Spesiale dierestudies het egter bewys dat daar verskille in lewensduur bestaan by nou verwante diere met verskillende genetiese konstitusies wat onder dieselfde omstandighede lewe.

Diere van verskillende spesies is gebruik om te bepaal wat die verhouding van groei en ontwikkeling tot lewensduur is. In hulle studies het navorsers toetse gedoen om te bepaal of die bevordering of vertraging van groei deur spesiale diëte 'n langer lewensduur in die hand werk of nie. Die rol wat groei speel in verband met lewensduur het 'n strydvraag geword. Die teorie wat die meeste byval vind is dat groei en langlewendheid nie onderling afhanklik is nie maar dat hulle nou verbonde is met die algemene metabolisme-tempo; hulle mag dus newegeskikte verskynsels wees onder beheer van dieselfde metaboliese beginsels.

Aangesien die leeftyd gewoonlik deur siekte beëindig word, moet ons die probleem van lewensduur benader met die oog hoofsaaklik op die faktore wat betrekking het op die bevordering van gesondheid of voorbehoeding van siekte, en op die bespoediging of vertraging van veroudering en van groei.

Beide onder- en oorvoeding het 'n nadelige invloed op die verloop van siekte, maar die verhouding van dieet en groei tot lewensduur is ietwat meer problematies. Dadelik doen 'n moeilikheid hom voor: wat is die optimale of normale dieet? Dit is twyfelagtig of 'n besondere dieet dieselfde uitwerking sal hê op groei, op reproduksie en op langlewendheid. Elke indiwidu het per slot van sake besondere behoeftes. Daar is ander voor die hand liggende moeilikhede. Voedsel-inname wissel volgens die seisoene en met die kwaliteit van die dieet; sintetiese of gesuiwerde diëte is bv. proefondervindelik nie so doeltreffend soos natuurlike voedsel nie. In laboratoriumstudies is die soort dier wat gebruik word, geslagsverskille, die hittegraad van die laboratorium, die effekte van bymekaarskaring of die veranderings in eetgewoontes as gevolg van enkelhokke, almal belangrike oorwegings wat in ag geneem moet word by toetse vir lewensduur. Natuurlik word die saak nog meer bemoeilik wanneer die waarnemings oor baie maande of selfs jare strek.

Dit is nouliks te betwyfel dat dieet-inkorting gedurende sekere lewenstye sommige proefdiere goed

certain periods of life has a beneficial effect on the animal; but it remains to be determined at what age the optimum effect can be got from this and whether the results obtained apply to mammals in general.

A high intake of protein or carbohydrate in the diet apparently does not militate against longevity provided there are no secondary deficiencies, but much fat in the diet has as a rule adverse effects on the life span. Laboratory experiments are here again not without special difficulties; for example, rats given a high-protein diet need more calcium and, unless this is provided, calcium deficiency enters into the picture. The life span of adult rats is unaffected by a diet with a protein content varying from 8 to 30%. In white mice receiving nucleic acid in addition to their basal diet the life span was prolonged, which may be interpreted as indicating that, from the point of view of longevity, the stock diet was made more nearly optimal by the supplement. The whole problem of the effects of different levels of dietary protein on length of life is far from being solved.

A well-documented review[1] has recently been published of the materials and methods used in nutritional experiments on the life span of animals and the effects of modifications of the diet on longevity. It is evident from the results so far available that much more work remains to be done on the effects of diet on life span.

1. Silberberg, M., and Silberberg, R. (1955): Physiol. Rev., **35**, 347.

doen, maar dit moet egter nog vasgestel word op watter tydstip die grootste voordeel hierdeur verkry word en of die uitslag van hierdie eksperimente van toepassing is op alle soogdiere.

'n Groot inname van proteïen of koolhidrate in die dieet het blykbaar nie 'n nadelige uitwerking op langlewendheid nie mits daar geen sekondêre tekorte is nie. Baie vet in die dieet het egter gewoonlik nadelige uitwerkings. Ook hier kom laboratoriumproefnemings voor spesiale moeilikhede te staan: rotte wat bv. 'n proteïenryk dieet hou het meer kalsium nodig, en tensy dit voorsien word tree 'n kalsiumtekort in. Die lewensduur van volgroeide rotte word nie beïnvloed deur 'n dieet wat tussen 8 en 30 persent proteïen bevat nie. Die leeftyd van wit rotte word verleng deur 'n byvoeging van nukleïensuur by die gronddieet. Daarvan kan ons aflei dat die gewone dieet baie meer voordelig gemaak word deur die byvoegsel. Die uitwerkings op die lewensduur van verskillende hoeveelhede proteïen in die voedselplan is 'n probleem wat nog lank nie opgelos is nie.

'n Goed gestaafde oorsig[1] het onlangs verskyn omtrent die materiaal en metodes wat gebruik was in voedingeksperimente aangaande die lewensduur van diere, en die uitwerkings van dieetwysigings op langlewendheid. Uit die uitslae tot dusver beskikbaar blyk dit duidelik dat nog baie werk nodig is op die gebied van dieet en sy uitwerking op lewensduur.

1. Silberberg, M. en Silberberg, R. (1955): Physiol. Rev., **35**, 347.

SUBCUTANEOUS FAT NECROSIS AND SCLEREMA NEONATORUM

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For many years these two conditions have been confused and their titles even interchanged, and though many articles have been written which differentiate them there is still a lack of universal agreement as to whether they are separate entities or not. The purpose of this paper is to attempt to clarify the matter; the position will be examined by a consideration of personally observed cases and a review of the available literature.

The essential clinical lesion in these states is a superficial tissue induration, and two other terms which have been used in this connection in the newborn period should be mentioned at this stage in order to clear the air for the main subject. These terms are Scleroedema and Scleroderma.

The name scleroedema has been used for the tense oedema which may occur in feeble babies, large or small. They often die, and the only abnormal histological finding in the subcutaneous tissue is intense oedema. It would probably be better to dispense with the term scleroedema altogether, for it does not appear to be a pathological entity in its own right.

Scleroderma, one of the collagen diseases, is rare in infancy. The skin is hardened and may later atrophy. Other tissues in the body may also be affected. A case recently described as sclerema neonatorum would, from the histological picture, appear more likely to have been one of this condition (Brescia and Tartaglione[1]). Perhaps one should not attempt to draw too fine a distinction between the two states, which may, in fact, be closely related.

Historical

According to McIntosh *et al.*[2] sclerema is an Italian form of the Greek word scleros (hard), and the first adequate description of the disease is attributed to Underwood in 1784. Still[3] gives Underwood's description of the condition as 'a peculiar tightness and hardness of the skin over almost the whole body. The skin instead of lying loose and pliable, is rigid as if it adhered to the bones....some children have been born with the complaint, none of whom have been known to live'.

Henoch,[4] in 1889, said that sclerema 'is a complaint about which, until recent times, there prevailed a greater confusion of opinion than almost any other disease'. He described the tense induration of the body 'as if it had been frozen' and mentioned that the disease is accompanied by a considerable fall of temperature in feeble premature infants, who invariably die. He quoted Parrot as saying that a French physician named Andry

created confusion by using the term sclerema for a case of oedema of the newborn and that Bouchut, another Frenchman, made matters worse by connecting sclerema with scleroderma, although 'they have nothing in common'.

The recognition of subcutaneous fat necrosis came much later. In 1907 Fabyan[5]: from the Johns Hopkins Hospital, reported the case of a baby with generalized induration of the subcutaneous tissues who was found accidentally suffocated in his cot. Material taken for section at the autopsy showed necrosis of the subcutaneous fat. Other names have been given to the condition, such as adiponecrosis subcutanea necrotica (Fox[6]), ischaemic fat necrosis (Farr[7]) and traumatic fat necrosis of the newborn (De Vel and Bolin[8]). These cause no difficulty, but when the term sclerema neonatorum is applied to the disease confusion arises.

Gray[9] writing in 1933 approved the description given by Ballantyne in 1895 of two conditions associated with hardening of the subcutaneous fat. The first, which he called sclerema neonatorum, consisted of progressive hardening of the fat in sharply defined patches in healthy babies. The second type of hardening was said to be rare. It was seen in cases of exhausting disease such as diarrhoea, came on quite rapidly, and affected the whole body. This second was a terminal state and was given the name of preagonal induration.

One would agree with Gray's statement that the histology in cases called by others subcutaneous fat necrosis was the same as in his cases of 'sclerema' because it is clear that he was describing what would nowadays be called subcutaneous fat necrosis. The term sclerema might be used for Ballantyne's preagonal induration.

In Tables II and III representative cases of subcutaneous fat necrosis and of sclerema neonatorum, respectively, are summarized.

SUBCUTANEOUS FAT NECROSIS

This takes the form of lumpy, indurated areas which are attached to the skin but not, as a rule, to the deeper structures. Their size may vary from that of a large pea to areas covering most of the back and buttocks. These sites, as well as the thighs, shoulders and cheeks, are the parts most likely to be affected. The skin tends to be slightly elevated and to become purplish-red over the affected parts, which may or may not be painful to the touch. Not infrequently the 'lumps' soften and liquefy after a week or two. In other cases there is no softening and the lumps merely fade away. Healing occurs in a few months, sometimes in association with calcification in the soft tissues. In one case described by Holzel[10] atrophy of the subcutaneous fat resulted, but this is unusual.

The affected babies are healthy full-term infants who have no difficulty with temperature control. The average time of onset is at about the end of the 1st week, though the condition has been seen in some cases soon after birth. In others again it has been reported when the baby was several weeks old. The prognosis is good and most babies recover, though there is no specific treatment which accelerates recovery.

Most authors mention that the baby had been born after a prolonged or difficult labour and that resuscitation has usually been necessary. This has been the experience of the present writer in the cases to be described. As regards the mechanism of the disease, De Vel and Bolin[8] have suggested that ischaemia follows compression of the babies' soft tissues either during labour or from over-vigorous attempts to resuscitate them after birth. Nowadays one would scarcely expect the latter to apply. Farr,[7] in order to see the effects of trauma, applied tissue forceps to the skin of pigs; 26 days later he excised the traumatized tissues and found histological evidence of fat necrosis. He mentions several cases in adult human beings where fat necrosis followed local trauma and he too considered that ischaemia is the cause of the condition. One occasionally sees babies with nodules in the neck or on the face after forceps delivery. These are presumably local areas of fat necrosis; but one has not felt that biopsies were justified to prove this. Lightwood[11] has also described cases with local lesions only.

Zeek and Madden[12] and Flory[13] claimed that it was not the subcutaneous fat alone which was affected. In each report a case was described in which necrotic changes were found in the fat around internal organs in the abdomen and thorax. They suggested, therefore, that obstetric trauma could not be the sole cause of the condition and Zeek and Madden attributed the lesions in their case to some generalized disturbance of fat metabolism which so changed the fat that it acted as a foreign body which initiated the typical changes. To examine this claim autopsy material is necessary, and as the affected babies almost always recover this is not easily come by. For the present, therefore, the exact cause of this condition must remain undecided.

CASE REPORTS

Case 1. The parents of baby C. noticed lumps in the skin soon after her birth. She was born in 1952 at full term after a difficult labour and required resuscitation. Generalized convulsions occurred during the first week. At the age of 7 weeks she was admitted to hospital suffering from gastro-enteritis and it was seen that there was hardening of the cheeks, arms and legs, with softer lumps on the back. The report on a piece of affected subcutaneous tissue from the calf, sent for histological examination, was that there were foci of fat necrosis with calcification and surrounding fibrosis. Some degree of softening had occurred when she was discharged at the age of 3 months. The mother has recently told me that return of the soft tissues to normal was not complete for nearly 2 years. The baby is mentally defective.

Case 2. Baby M. was born at term after a precipitate labour, and also needed assistance after birth. Induration of buttocks and upper thighs was seen on the 5th day. Largely as the result of a pathological report on a piece of tissue sent for examination, a diagnosis of sclerema was made and treatment with ACTH commenced, 5 mg. being given twice daily. A note was made to the effect that the baby did not look ill and was feeding well. Softening was evident on the 8th day and on the 11th day creamy fluid was aspirated. Microscopic examination showed fat crystals and 'large cells'. On discharge from hospital at the age of 1 month there were still large areas of induration present, though considerable softening had taken place. Recent Information is that the soft tissues did not feel normal until the baby was 7 months old. Death took place from malnutrition at the age of 16 months.

This is the only case of the series not seen by myself; the information came from the records of St. Monica's Home, Cape Town, and from the attending doctor who has kindly given permission for this case to be included. However, I saw the histo-

logical specimen, and this showed the typical changes of fat necrosis. It is clear that this was the correct diagnosis, which is supported by the fact that hormone treatment had no effect on the course of the disease.

Case 3. Baby V. was admitted in June 1953 aged 16 days, with signs suggesting septicaemia. He had been delivered by forceps and was said to have been very weak at birth. At 6 weeks, when recovery from the septicaemia was well advanced, induration of shoulders and buttocks was seen. The hardened tissues could be moved over the deeper parts. The infant was discharged in good health 10 days later and a recent report from the mother is that return of the superficial tissues to normal was complete by the age of 6 months. The baby is now completely well except for a stiff ankle.

Case 4. Baby P. Here the obstetric and neonatal histories were normal. Thickening of buttocks and scapular regions was noticed on the 3rd day, the skin over these parts later becoming a purplish-red colour. The general condition of the baby was very good. Spreading of the induration to the shoulders and thighs followed and the baby resented being handled. A biopsy specimen was taken and showed extensive fat necrosis. Despite some delay in healing of the site where the biopsy had been made, the baby was discharged well in June 1954, aged 2 months, but hardening was still present. When seen again in May 1955 the baby was in good health, the mother stating that the affected tissues had become normal not long after the return from hospital.

Case 5. Baby A. was born in July 1954, after a normal labour with no difficulty after birth. On the 5th day a hard knob, the size of a cherry, was noticed on the outer side of the left upper arm and over it there was slight discolouration of the skin. The swelling was attached to the skin but could easily be moved over the underlying muscle. The baby was not in any way incommoded and on discharge, aged 13 days, the lump was getting smaller. This is an example of localized fat necrosis.

Case 6. Baby T. This was the most recent case, the baby having been born in March 1955 at full term after a difficult forceps delivery. It did not suck for some weeks and vomited for the first fortnight. The limbs were seen to be stiff from the 2nd day, and induration in the thighs, buttocks, back and upper arms was palpable at the same time. The next day the skin over the shoulders and buttocks was purplish-red. There were areas of softening present by the 2nd week and the induration now involved the cheeks, calves and forearms. Fluid aspirated from one of the fluctuating areas showed the typical picture of sheaves of needle-like crystals, macrophages and fat globules. At 2 months some induration was still present, though the baby was otherwise well.

SCLEREMA NEONATORUM

This condition, which is much rarer than subcutaneous fat necrosis, also comes on at about the end of the 1st week of life but, as in fat necrosis, cases are sometimes seen within a day or two of birth. Some writers have stated that it is a disease of premature babies (Potter[14] and Grotts[15]), while others, including Hughes and Hammond[16] in a review of the condition, found only a small percentage of the babies to have been premature. Of the cases to be reviewed in this article about half the babies were premature.

Induration commences especially in the legs and cheeks and is liable to spread to the whole body with the exception of the palms, soles and genitalia. The overlying skin, which does not pit on pressure, looks white or mottled and feels cold and very hard. It cannot be picked up on the subcutaneous tissues, and the latter in turn appear to be adherent to the deeper structures below. The limbs may be rigid as a result of the hardening and if large areas are involved there may be but little spontaneous movement. The indurated parts do not appear to be painful.

Before the introduction of cortisone and ACTH the disease was in most cases fatal within a few days. In 5 cases where recovery was reported before treatment with these hormones was available (Paterson,[17] Bourne,[18] Nash,[19] Allyn and Marek,[20] and Hughes and Hammond[16]) the main form of treatment was heat, either as hot baths or by incubator. More frequent recoveries have been reported under treatment with ACTH and cortisone (Kendig and Toone,[21] Kendall and Ledis,[22] Williams,[23] and Eisenoff *et al.*[24]). ACTH has usually been given in doses of 5 mg. 6 or 8 hourly for several days and cortisone in 10 mg. doses at similar intervals.

On section of the superficial tissues the skin is reported to be normal in all respects and there is no escape of fluid from the subcutaneous layer. The latter is thicker and the fibrous trabeculae are larger than normal. In the opinion of some this thickening is the essential lesion of the condition. The response to ACTH and cortisone would, theoretically, fit in with this conception. Other writers, however, report that there are no changes in the subcutaneous tissues. Despite this, there seems to be general agreement that no giant cells or crystals are found to suggest fat necrosis (Fox,[6] Nicholson and Claireaux,[26] Potter,[14] and Holt and McIntosh[25]).

Fat consists of a combination of glycerol and the fatty acids oleic, palmitic and stearic. Oleic-acid fat is liquid at low temperatures while palmitic-acid and stearic-acid fats are solid even at body heat (Best and Taylor[27]). The higher the proportion of oleic acid the softer the fat is likely to be. McIntosh *et al.*[2] quoted Langer, who found in 1881 that the proportion of oleic-acid fat in adults was 86% while in newborn infants it was only 65%.

Channon and Harrison[28] reported a study of subcutaneous fat in normal and scleremátous infants. In the first place they found that the melting point of fat in young infants was higher than in adults. On this point there was already general agreement. Their second finding was that the melting point of the fat in 4 scleremátous babies in their tests was higher than in the normal babies. They wondered if this indicated a lower oleic acid content in sclerema, but they could not find any direct evidence of this. This work does not seem to have been confirmed nor is one completely satisfied regarding the criteria for diagnosis of their cases of 'sclerema'.

As histology shows no evidence of fat necrosis or inflammatory infiltration, neither trauma nor infection would appear to be likely causes of the condition.

Hughes and Hammond[16] examined the possibility, already accepted by some, that sclerema neonatorum might result from the combined effect of the low proportion of oleic-acid fat in infants and chilling of the body. They state that the actual melting points of infant fat would seem to rule out lowered body-temperature as a likely factor. In any event not all babies with sclerema have low temperatures. A point, however, was made of the fact that it is not the mouth or rectal temperature which matters but that of the peripheral tissues, and that the two may be different. Hughes and Hammond went on to develop the interesting theory that shock may be the precipitating factor in sclerema. They stated that in most cases there is evidence of preceding disease of one kind or another which may initiate a state of shock, this in turn resulting in peripheral circulatory failure which reduces the peripheral temperature to such an

extent that cell metabolism is affected. This in some way enhances the hardening of fat in the newborn, probably as a result of the alteration of connective-tissue collagen. The condition would then come into the group of collagen stress-diseases and the reports of success with ACTH and cortisone would appear to lend support to this conception.

Case Report

In May 1953, a baby was admitted to hospital on the 2nd day of life because of persistent vomiting. This proved to be due to intestinal obstruction from meconium ileus and operative treatment was undertaken for its relief. On the 7th day generalized induration of the trunk, limbs and cheeks was noticed. The skin showed the marbled appearance often described in sclerema and the indurated areas could not be moved on the structures lying beneath them. Cortisone was commenced, 10 mg. being given twice daily for 4 days until the 11th day, when the baby died from a surgical complication. There was already considerable generalized softening of the indurated areas before death took place. A piece of abdominal skin and subcutaneous tissue removed at autopsy was recently re-examined but it does not show any convincing alteration of the connective tissue. At the same time, there is no evidence of fat necrosis. Whether the treatment with cortisone had already restored the tissues to normal is a matter for conjecture only, but it does not seem likely.

DISCUSSION

At this stage it may well be asked whether, in fact, subcutaneous fat necrosis and sclerema neonatorum are separate entities or merely the same disease in different degrees of severity.

There are a number of points in common, such as lumpy induration of the subcutaneous tissues in more or less the same parts of the body, coming on at about the same time in the neonatal period in each case, commonly with a history that the baby was weak at birth.

The points of difference, however, are even more in number, as is shown in Table I. In fat necrosis the babies are nearly always fully mature whereas about 50% of those with sclerema are premature babies. The purplish-red colour of the skin and the tendency to liquefaction are seen only in subcutaneous fat necrosis. The point that the indurated tissues in fat necrosis are movable on the deeper structures while in sclerema they are not, is a matter of importance, though it must be admitted that in very extensive fat necrosis there may sometimes be difficulty in establishing this difference. Prognosis and treatment of the two states also reveal differences, recovery without treatment being the rule in subcutaneous fat necrosis while in sclerema neonatorum death is almost certain unless ACTH or cortisone is used. This treatment can be life-saving in sclerema, but in fat necrosis it is quite without effect. The histology is another point of essential difference, there being evidence of fat necrosis in the one case and not in the other. There may in addition be some collagen change in the sclerematous patient.

TABLE I

	Sclerema neonatorum	*Subcutaneous Fat Necrosis*
Gestation	About 50% premature	Full term
Labour	Usually normal	Usually long or difficult
State at birth	Mostly weak or cyanosed	Mostly needed resuscitation
State at onset	Weak or debilitated	Healthy babies
Date of onset	Average 9th day (26 cases) extremes 1 to 70 days	Average 7th day (16 cases) extremes 1 to 42 days
Sites	Whole body except palms, soles and genitalia	Buttocks, shoulders, thighs, back, cheeks, separately or together Seldom as universal as sclerema
Attachment	Skin and deep structures	Skin only, usually movable over deep structures
Histology	Thickening of subcutaneous connective tissue, or *nil* abnormal. No fat necrosis	Fat necrosis with foreign-body giant cells, macrophages, and fatty acid crystals
Treatment	ACTH or cortisone	None
Outcome	Before ACTH and cortisone mostly died. Now most recover if treated	Recovery usual
Cause	? stress disease	? trauma

In the light of the preceding remarks there would seem to be sufficient justification for believing that subcutaneous fat necrosis and sclerema neonatorum are two different diseases.

As regards aetiology there would seem to be quite a good case for sclerema neonatorum being a stress disease affecting collagen in the subcutaneous tissues. Although the skin is not affected in sclerema it is possible that it is closely related to scleroderma. It may be that they are variants of the same disease.

In subcutaneous fat necrosis several questions remain unanswered. Birth trauma is evident in most cases but not in all. What causes the condition in the latter cases? If the process of birth is the precipitating cause why is the onset in some cases much later than in others? How can one account for the fact that in 2 reports quoted necrosis occurred in fat around internal organs as well as in the subcutaneous tissues? It is possible that trauma is only one factor in the initiation of the condition and that there is some further cause operating which is as yet unrecognized.

SUMMARY

The essential lesion in subcutaneous fat necrosis and sclerema neonatorum is induration of the subcutaneous tissues in the newborn period and these conditions are discussed and compared.

An account of the history of these affections is given to show how they have been confused with each other in the past and in an attempt to clarify the position a description of each condition is given together with illustrative cases. These are coupled with other cases appearing in the literature.

It would seem clear that there is a difference between the two conditions and this is of more than academic importance because in subcutaneous fat necrosis the infants recover whether treated or not; treatment, in fact, having no effect on the speed of recovery. On the other hand, in sclerema neonatorum, admittedly a rare disease, the babies nearly always die unless the correct treatment is given.

While sclerema neonatorum is probably a stress disease affecting subcutaneous tissue collagen, the cause

TABLE II. SUBCUTANEOUS FAT NECROSIS. REPRESENTATIVE CASES

Author		*Birth*	*State after Birth*	*Onset*	*Histology*	*Outcome*
Fabyan[5]		Normal	Needed resuscitation	3 day	Fat crystals, giant cells, mononuclears	Suffocated in cot 14th day
Farr[7]		Very difficult	Not stated	2 day	Hard grey-white fat, crystals, giant cells	Nearly all gone at 2 months
De Vel and Bolin[8]		Low forceps	Slight asphyxia	14 day	Fan and rosette shaped crystals, giant cells, macrophages	Gone 1½ months after onset
Zeek and Madden[12]		Long labour	Cyanosis, arms paralysed	9 day	Needle crystals, F.B. giant cells, macrophages, fibrous tissue proliferation. Same changes in fat around organs in thorax and abdomen	Found dead in cot 16th day
Flory[13]		Long labour	Normal	2 day	Necrosis of subcutaneous fat also that around kidneys and ribs	Died at 2 months. Gastro-enteritis
Lightwood[11]	1.	Long labour	Normal	Birth	Fat necrosis, F.B. giant cells (local lesion)	Gone about 1½ months after onset
	2.	Normal	Normal	3 day	Not given. (Local lesion)	Almost gone at 2 months
Holzel[10]	1.	Normal	Needed resuscitation	2nd week	Fatty acid crystals, histiocytes, giant cells, occ. polymorphs and lymphocytes	Clear at 2 months. Small area fat atrophy
	2.	Normal	Cyanosis, atelectasis	2nd week	Not given	Clear by 2 months
	3.	Difficult forceps	Severe asphyxia	12 day	Not given	Clear by 2 months
Present cases	1.	Difficult labour	Very weak. Fits first 4 days	Birth	Fat necrosis, giant cells, calcification fibrosis	Still marked induration when discharged at 3 months. Clear at 2 years
	2.	Precipitate labour	Resuscitation needed	5 day	Fluid aspirated showed fat crystals, large cells. Biopsy, fat necrosis	Clear by 7 months
	3.	Forceps	Very weak. No cry or sucking 1st 3 days	6th week	Not done	Unchanged when discharged 10 days after onset. Clear at 6 months
	4.	Normal	Some cyanosis	3 day	Extensive fat necrosis	Clear before 3 months
	5.	Normal	Normal	5 day	Not done (local lesion)	Clearing on discharge 13th day
	6.	Difficult forceps	Weak, no sucking 1st 2 weeks	2 day	Fluid aspirated showed fat crystals, macrophages, fat globules	Clearing but still some induration at 2 months

TABLE III. SCLEREMA NEONATORUM. REPRESENTATIVE CASES

Author		*Gestation*	*State after Birth*	*Onset*	*Temperature*	*Histology*	*Treatment*	*Outcome*
Hughes and Hammond[16] Review up to 1948*		11 full term, 4 premature	Two-thirds feeble or cyanosed	Average 8th day	10 subnormal 4 fever	Increase in connective tissue in 5 of 10 Examined.	Non-specific, chiefly heat	14 died out of 19
Kendig and Toone[21]		Premature	Feeble, cyanosed	2 day	Subnormal	Not reported	Cortisone 8 hourly 1st 24 hours, then 12 hourly for 3 days	Recovered
Grotts[15]		Premature	Difficulty with respiration	17 day	Subnormal	Not reported	No hormones	Died
Kendall and Ledis[22]		Premature	Difficulty with respiration	2 day	Normal	Not reported	ACTH 6 hourly 8 doses	Recovered
Williams[23]		Premature	Normal	2 day	Subnormal	Not reported	ACTH 12 hourly 3 days, then once daily for 2 days	Recovered
Eisenoff *et al.*[24]	1.	Full term	Normal	6 week	Subnormal	Not reported	ACTH 6 hourly for 12 doses	Recovered
	2.	Premature	Normal	3 day	Normal	Not reported	ACTH 8 hourly for 4 days	Recovered
Present case		Full term	Weak; meconium ileus	7 day	Normal	No connective tissue changes. No fat necrosis	Cortisone 12 hourly for 4 days	Died, operative complication

* Nineteen cases in all. Full details not given in all cases.

of subcutaneous fat necrosis, thought by some to be due to birth trauma, is at present not completely understood.

Some of the babies were seen at Groote Schuur Hospital and others at St. Monica's Home, Cape Town. My thanks for the use of clinical material are due to Professors F. J. Ford and J. H. Louw of Groote Schuur Hospital, and to Dr. E. Barrow, Honorary Superintendent, St. Monica's Home; also to the Pathology Department, University of Cape Town, for their reports and for allowing me to examine the sections.

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POSITIVE-PRESSURE RESPIRATORS IN THE TREATMENT OF POLIOMYELITIS

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The commonest cause of death in poliomyelitis is respiratory failure. Patients with poliomyelitis of the spinal type who develop respitatory failure have for many years been successfully treated with tank respirators (the 'iron lung'). In bulbo-spinal poliomyelitis, however, where in addition to respiratory failure there is a loss of the cough and swallowing reflexes, treatment in a tank respirator frequently leads to the inhalation of secretions and vomitus, with a fatal result.

The work of Lassen [1] during the severe polio epidemic in Copenhagen in 1952, in which a high proportion of the cases were of the bulbo-spinal type, has demonstrated that the method of choice in these cases is positive-pressure respiration through a tracheostome. The technique used by the Danish workers is diagramatically represented in Fig. 1. The re-breathing bag was manually compressed rhythmically, by relays of medical students working in 8-hour shifts. At one time 70 patients were being mantained in this manner at the Blegdam Hospital. Since then various types of mechanical positive-pressure respirators have been introduced.

This method, while overcoming the respiratory failure from which the earlier patients died, introduced hazards resulting from the effects of positive-pressure respiration on the circulation. Patients that died during the Danish epidemic did so not from respiratory failure, but from circulatory failure (Astrup *et al.*[2]).

During the recent poliomyelitis epidemic in Natal it was felt desirable to acquire a number of positive-pressure respirators in order to be able to treat bulbo-spinal poliomyelitis after the method of Lassen. Naturally, the type of respirator which was least likely to produce deleterious effects on the circulation was desired.

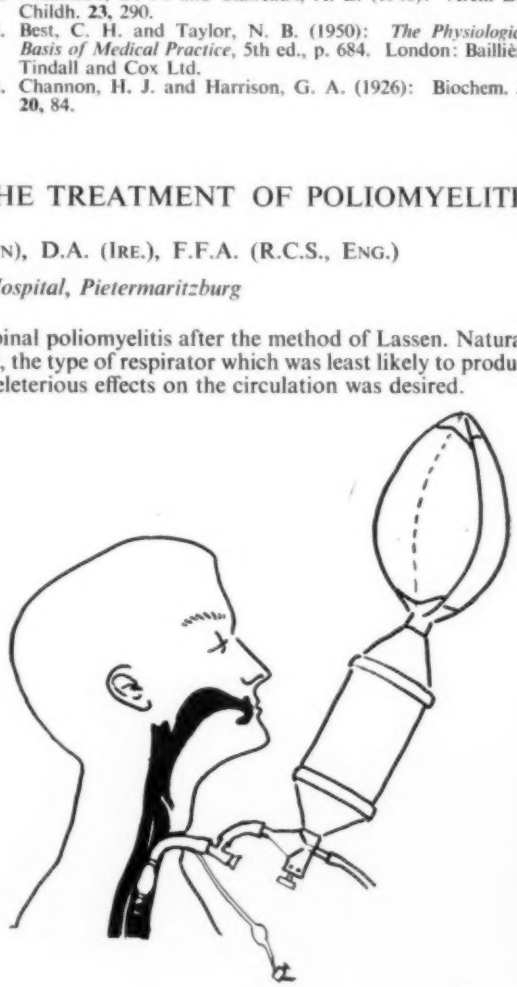

Fig. 1. Apparatus used by Lassen

The object of this paper is to compare a number of positive-pressure respirators, all of which are obtainable in this country. In particular, comparison has been made of the respiratory pattern which each produce; and thus an assessment attempted of their probable effect on the circulation.

Positive-pressure respirators may be divided into the pump type and the pressure-sensitive type (Mushin and Rendell-Baker.[3, 4] In the pump type a predetermined volume of air and/or oxygen is pumped into the lungs irrespective of the pressure produced (within safe limits). In the pressure-sensitive type the lungs are inflated until a predetermined pressure is reached, which then cycles the machine, irrespective of the volume which has been delivered to the lungs.

Criteria of a satisfactory machine are as follows:

1. The inspiratory phase should be rapid and the inflation pressure should be variable between 5 and 30 cm. H^2O.
2. During the expiratory phase this pressure must drop very rapidly to atmospheric or below.
3. Expiration must begin immediately the maximum pressure has been reached. The lungs must not be held inflated. In all pump-type respirators there tends to be a lag between the end of inspiration and the beginning of expiration, producing a plateau effect on the pressure curves.
4. The rate should be continuously variable, so that the machine can be synchronized with the patient's own efforts. The machine must not act in defiance o the patient's own efforts.
5. The minute volume delivered by the machine should be measurable.
6. A safety valve should be incorporated, and the machine must be mechanically sound and capable of continuous use without breakdowns.

METHOD IN THIS STUDY

Various types of positive-pressure respirators were acquired. Some of these are designed primarily for use on apnoeic patients during anaesthesia, others are designed primarily for the treatment of respiratory failure resulting from poliomyelitis or other causes. For the comparison, anaesthetized patients were used in whom spontaneous respiration had been abolished with curare. Pressure tracings were then taken from the endotracheal mount, using a tambour and kymograph.

1. *The Aintree Respirator* (Esplen[5]). This machine is designed primarily for use in anaesthesia. Fig. 2 is a pressure tracing from it. As will be seen, it employs positive pressure only, and on our recording apparatus the pressure never fell to atmospheric level during expiration. We treated one patient with bulbo-spinal poliomyelitis and complete respiratory paralysis with this machine. The patient's condition was excellent for the first 24 hours but thereafter he developed signs of progressive circulatory failure and died after about 48 hours. This may have been due to the effects of positive-pressure respiration on the circulation. The machine, however, is excellent for the management of apnoea during anaesthesia. It incorporates an automatic expiratory valve which conveniently deals with an excess flow of anaesthetic gases. It is supplied with an electric air compressor.

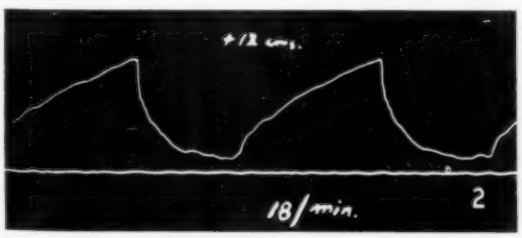

Fig. 2. Pressure curve from Aintree respirator

2. *The Pask Respirator* (Pask[6]). This was designed as an emergency respirator of simple construction. A model was built by a local engineering firm. As will be seen from Fig. 3, the respiratory pattern it delivers is a very satisfactory one. Both the inspiratory and expiratory phases are rapid, and there is an adequate pause. Although there is no negative phase this machine gave the most satisfactory respiratory pattern of all the positive-pressure respirators we have used. It may be run on an electric air-compressor or on a cylinder of compressed air.

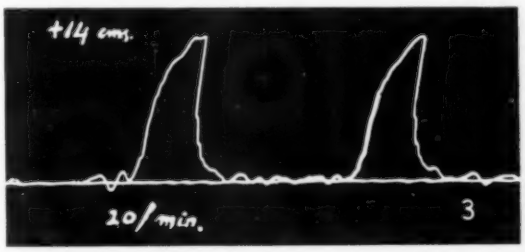

Fig. 3. Pask respirator

3. *The Poliomat* (*Drager*). This very compact respirator supplies both positive and negative pressure. Fig. 4 represents a pressure tracing from it. The negative expiratory pressure is about half the positive inspiratory pressure. It is pressure-sensitive, and both the rate and the pressure are continuously variable. The minimum pressures are +9 and —4 cm. H^2O, which are possibly somewhat high. There is no means of measuring the

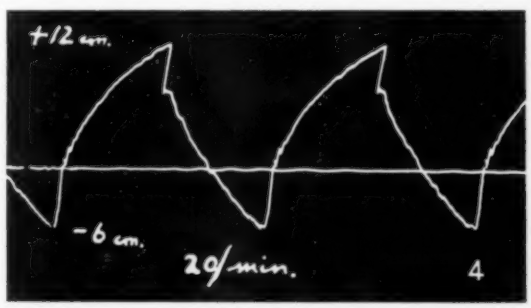

Fig. 4. Poliomat respirator

tidal volume delivered to the patient. The machine incorporates a humidifier, which utilizes the water vapour of the exhaled air; however, we are somewhat sceptical of the efficiency of this humidifier. Using a hair hygrometer we found the relative humidity of the inspired gases to be 70% at room temperature after 30 minutes' use. The machine runs on a cylinder of oxygen and delivers about 50% oxygen to the patient. The oxygen consumption is high. We have been most impressed with the performance of this respirator.

4. *The Pulmomat* (Drager). This machine is similar to the Poliomat but is designed primarily for use in anaesthesia. The pressure curve is similar to that of the Poliomat. There is no automatic expiratory valve such as is found on the Aintree respirator. Consequently, if the machine is used with a semi-closed circuit and a flow rate of say 2 litres per minute, it is essential to incorporate a manometer; otherwise it is impossible to adjust the expiratory valve so as to balance the positive and negative phases of the cycle. Under such circumstances, moreover, the tidal volume will be less than that registered by the machine.

Other Machines. Numerous other machines have been described in the literature. Astrup *et al.*[2] have found the Engeström respirator[7] the most satisfactory machine they have used; the price of this machine in South Africa is high (about £1,200). They also recommend the Bang respirator.[8] Machines similar to the Bang are the Clevedon respirator[9] and a machine described by Pask.[10] We have not used the Radcliffe respirator (Russell and Schuster,[11] which employs positive pressure only. The peak inspiratory pressure tends to be prolonged, as can be seen from the pressure curve in a paper by Smith *et al.*[12] The control of the respiratory rate is not ideal. The advantages of this machine are that it can use room air, oxygen or any other gases, and the minute volume delivered to the patient can be measured.

DISCUSSION

The institution of positive-pressure respiration may have profound effects on the circulation, and physiologists have been aware of this for some time (Moore *et al.*[13]) The most marked effect is a fall in cardiac output (Humphreys *et al.*,[14] Otis *et al.*[15]) The cause of this fall in cardiac output would appear to be the abolition of the negative intrapleural pressure which is present during spontaneous respiration and which plays an important part in the maintenance of the venous return to the heart. Other sequelae that have been noted with pressure respiration are a fall in systolic and diastolic blood pressure (Beecher *et al.*[16]) and a rise in central and peripheral venous pressure (Carr and Essex[17]).

The effects of positive pressure respiration on the circulation have been shown to be very much more marked in the presence of haemorrhagic shock (Carr and Essex[7]). Maloney *et al.*[18] have also demonstrated that whereas with a normal circulation the effects of positive pressure respiration are negligible, after haemorrhagic shock or barbiturate poisoning the effects are profound.

Werkö[19] and Motley *et al.*[20] have shown that the fall in cardiac output is closely related to the mean pressure applied to the lungs. With any positive-pressure respirator, therefore, it is essential that the mean pressure applied should be kept as low as possible. In other words, the pressure curve must conform to a certain pattern. It should show a moderately rapid rise to a maximum during inspiration, followed by a very rapid fall to zero pressure during expiration. There should

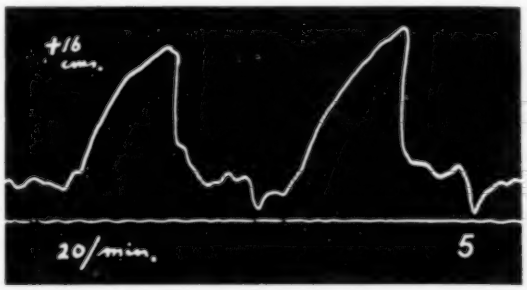

Fig. 5. Manual compression of re-breathing bag

then follow a pause, at least as long as the period of inflation. The rise in intrathoracic pressure is small, and the cardiac output is unaffected (Cournand *et al.*[21]) Such a pressure curve we have obtained with the Pask respirator (Fig. 3). A similar curve may be obtained by manual compression of an anaesthetic bag, but it will be found that it frequently does not fall to zero during expiration (Fig. 5).

Werkö[19] has stressed the importance of the pressure during the expiratory phase of pressure respiration. The expiratory pressure gives a relatively higher pressure in the intrapleural space and influences the venous return to the heart more than does the inspiratory pressure.

Positive-pressure respirators may be divided into those which supply positive pressure only, the pressure falling to atmospheric during expiration (Fig. 3), and those which supply positive pressure during inspiration and a negative pressure during expiration (Fig. 4). Maloney *et al.*[18] have shown the importance of this negative pressure during expiration. With normal patients neither positive pressure alone, or alternating positive-negative pressure, have any significant effect on the cardiac output or blood pressure. In the presence of circulatory depression, however, respirators which employ positive pressure only were found to cause a marked fall in the cardiac output and blood pressure, sometimes of sufficient magnitude to threaten the patient's life. On the other hand the use of a respirator which supplied a negative pressure during expiration avoided this dangerous circulatory depression.

A comparison of the effects of positive pressure alone, and of alternating positive-negative pressure, on the intrapleural pressure is demonstrated by the oesophageal pressure tracing in Fig. 6. The oesophageal pressure was used as a measure of the intrapleural pressure-changes (Fervis *et al.*[22]) At A in Fig. 6 the patient was being respired with a positive-negative

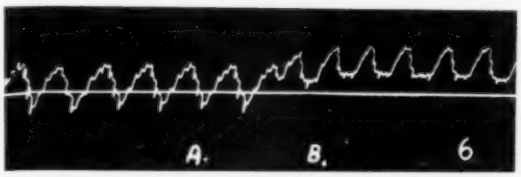

Fig. 6. Oesophageal tracing. A. Positive-negative respirator. B. Positive pressure only

respirator. At B he was changed over to positive pressure only. The marked rise that occurred in the intrapleural pressure is noteworthy.

In patients with poliomyelitis, circulatory depression may already be present as a result of asphyxia or from invasion of the vasomotor centre by the virus. If such a patient is now treated with a respirator which employs positive pressure only, the additional burden on the circulation resulting from this rise in intrapleural pressure may be of considerable importance.

It is desirable, therefore, that if respiratory failure is treated by the method that has been discussed, a respirator should be employed that delivers a negative pressure during the expiratory phase of its cycle. Alternatively, if positive pressure only is employed, the pressure curve should conform to that recommended by Cournand. The circulatory effects of positive-pressure respiration will then be minimized.

SUMMARY

A comparison is made of various types of positive-pressure respirators used for the treatment of respiratory failure from poliomyelitis and other causes. Anaesthetized patients in whom respiratory failure has been induced with curare were used for the purposes of the comparison. The pressure curves delivered by the machines were recorded by means of a tambour and kymograph. The effects of positive-pressure respiration on the circulation are discussed.

It is pointed out that if the circulatory effects of pressure respiration are to be minimized, it is essential to use a machine which delivers the correct type of pressure curve. The importance of a negative pressure during the expiratory phase is emphasized.

I wish to thank Dr. R. E. Stevenson, Director of Provincial Medical & Health Services, Natal, for his generous assistance, and Dr. S. Ripley, Senior lecturer in Physiology, Medical School University of Natal, for his invaluable advice.

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HEART DISEASE AND THE EFFORT SYNDROME

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The pain of angina pectoris is usually attributed to a relative myocardial ischaemia, and the demonstration of a coronary insufficiency, particularly with an exercise test in an individual giving the classical history, is taken to clinch the diagnosis. On the other hand when the typical asthenic individual develops an anxiety state, with a background of subconscious or conscious fear of heart disease, the effort syndrome results; it is fortunate that in the majority of such cases the over-all picture, to the trained observer, is very different from that of organic disease.

The effort syndrome may be called psychosomatic, since it originates with the emotion of anxiety or fear and thence *via* the hypothalamus and resultant autonomic imbalance the classical syndrome evolves, cloaked, fortunately for us, by symptoms (such as the inframammary ache) which the sufferer believes to be the correct outward manifestation of a disordered heart. It is usually easy, when assessing the symptoms, to differentiate the two conditions, particularly if the physical signs indicate the presence of a normal heart. A very different situation arises, however, when any cardiac disorder is present. Here again, anxiety and fear are present, but we often have a more stoical

individual than the constitutionally susceptible one. However, such factors are variable and the individual's 'threshold', above which autonomic imbalance will result from stress, will determine how much his symptoms *and signs* are organic and how much functional. Furthermore since he or she has experienced the correct symptoms of heart disease, often in a terrifyingly impressive manner, the history given is often indistinguishable from the real thing.

This must surely be the explanation of a dramatic reduction in the incidence of anginal pain, produced by the placebo, when remedies for angina are tested by means of the 'double bluff' technique.[1, 2] Similarly it accounts for the considerable improvement following a cardiac operation when all available methods of investigation demonstrate that no actual improvement in cardiac dynamics have been achieved. This constitutes 'psycho-surgery'. Soloff and Zatuchni [3] cite a remarkable case where a lady who for years had been confined to one floor of her house, and mainly to one chair on that floor, asked permission to seek outside work after an operation at which an auricular septal defect, too big to allow of any operative interference, had been found.

Even our scientific methods of investigation avail us little in sorting out the organic from the functional where organic disease already exists as a background. The electrocardiogram, for example, may be abnormal and an exercise test out of the question. An overlay of autonomic overaction may, in fact, be depressing an S-T segment further than would be the case were the subject less anxious. (In this connection it is worth while to remember that not infrequently the S-T segment becomes deeply depressed in a prolonged paroxysmal tachycardia where no subjective sensation indicating insufficiency is experienced.)

The point to bear in mind, then, is that the effort syndrome and true cardiac disease are not always separately occurring entities, but that the effort syndrome is probably most commonly encountered in the individual already suffering from heart disease. While it may then still preserve distinguishing features it may simply intensify and multiply what we regard as symptoms and signs of true cardiac disease. Perhaps, under these circumstances we simply recognize it as a 'psychological overlay' but nevertheless it remains a syndrome with an emotional stimulus involving the autonomic nervous system, though it is now modified by and sumperimposed upon a genuine cardiac insult. The result of this is that unnecessary iatrogenic disability occurs, not only in people with normal hearts, but also in those who have a cardiac lesion. In the latter case, indeed, since an organic insult has occurred, the soil is even more fertile.

It therefore behoves us always to bear in mind, for example, that the degree of dyspnoea in a case of mitral stenosis (and therefore the 'grade' of failure), or the number of anginal attacks in an individual with a coronary insufficiency, are probably exaggerated by the ultimate effects of anxiety. Not only is this important in determining a psychotherapeutic attack on the disability, and the achievement thereby of a better mental state in the cardiac invalid, but also it must result in an improvement in his actual cardiac dynamics.

The effects of anxiety undoubtedly constitute a hazard to the cardiac invalid. Thus the labile hypertensive has a higher blood pressure during periods of stress (and it is interesting to note that Rauwolfia derivatives are now being successfully used for 'tension states' where no hypertension exists[4]). The hypocapnia of hyperventilation in the anxious individual produces peripheral vasodilatation and hypotension:[5] while the tachycardia common in the effort syndrome must surely affect the cardiac's metabolic economy, even where it results in a resting pulse rate of, say, 90, when in the absence of anxiety it might be 70. In this connection it is interesting to note that life-insurance experience indicates that applicants with the Wolff-Parkinson-White syndrome are not standard risks, their mortality being approximately 3 times the normal.[6] Similarly Bellet[7] mentions that a paroxysmal tachycardia, sufficiently prolonged, may cause irreversible damage and, when it is borne in mind that a large proportion of apparently normal people living on the diet of the Western world have significant coronary atheromatous change,[8] it becomes apparent that any tachycardia can seldom be contemplated as ultimately benign.

It is already the doctor's practice to sort out the functional from the organic symptoms and to deal with them by such psychotherapeutic methods as rationalization and reassurance, but when the functional effects are simply superimposed on the organic it becomes more difficult to discern them, and in fact it may sometimes be impossible. The suspicion of their presence is something that should always guide our approach to prognosis. Undue caution, as interpreted by the patient, is certain to entrench the functional overlay and yet, only too often, we find it being exercised. Rather should we err on the side of optimism, because by so doing we can put the patient through a psychotherapeutic 'test of cure'. It will be argued that this may at times be dangerous, and even disastrous, but in fact this should very rarely be the case.

On the one hand we have the case where there are plenty of symptoms and no signs, and on the other the case where our clinical and accessory methods of diagnosis confront us with impending disaster. In the far larger intermediate group we shall often find some little discrepancies if we search with a suspicious mind. It is here, with our reputation at stake, that we are tempted to cautious prognosis, but we must bear in mind that by giving advice in this sense we may initiate or entrench a functional overlay and that this will actually have physical, as well as mental, sequelae.

The crux of the matter, then, is that we must not always consider the effort syndrome and organic heart disease separately but rather must be careful that we do not superimpose the one on the other.

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RESEARCH FORUM, UNIVERSITY OF CAPE TOWN

ABSTRACT OF PAPER ON DIAMOX DIURESIS READ BY R. E. BERNSTEIN, M.B., B.CH., D.C.P., ON 5 OCTOBER 1955

Congestive heart failure is as frequent a cause of death as cancer. The rising tide of excess body-fluid is a major determinant in refractory failure. Mercurials, which suppress oxidative processes in renal tubules, cannot be regarded as safe and easily administered diuretics.

Carbonic anhydrase inhibitors (oral Diamox, Dirnate) offer a new approach to the therapy of cardiac oedema. Such drugs decrease the exchange of cellular H+ for Na+ from the tubular fluid, producing an osmotic diuresis of sodium and potassium bicarbonate.

Current clinical reports on these substituted sulphonamides indicate a difference of opinion regarding their efficacy. Our studies in healthy subjects with single doses revealed an increase in urinary pH, volume, sodium, potassium and bicarbonate, no effect on other ions and organic substances, and a decrease in ammonium excretion. The degree and duration of the increased electrolyte excretion and diuresis was related to the drug dose. The subsequent compensatory renal acidification with restoration of acid-base balance was slow.

Continued administration of Diamox produced a metabolic acidosis with a diminished diuretic response. Similarly, negligible diuresis occurred in subjects previously treated with NH_4Cl.

On a low-salt diet the major cation execreted after Diamox was potassium, with a negative potassium balance after repeated doses. This suggests that the possibility of potassium deficiency should be watched for in salt-restricted cardiacs given Diamox.

The limited diuretic effect of Diamox is related to the smaller amount of body bicarbonate compared with the action of mercurials on chloride execretion. Restoration of plasma bicarbonate must occur before Diamox can produce a subsequent diuresis. However, the acidosis created by prior Diamox administration will potentiate the effect of mercurials.

REPORT BY MEDICAL COUNCIL ON INQUIRY INTO CONDUCT OF A DOCTOR

At its meeting held in September 1955, the South African Medical and Dental Council considered a report of a special disciplinary committee on an enquiry into the conduct of Dr. N.G., held in Johannesburg on 18 and 19 April and 2 May 1955. The following charges were preferred against Dr. G.

'That you, being a medical practitioner registered under the Medical, Dental and Pharmacy Act, 1928 (Act No. 13 of 1928), as amended, are guilty of improper conduct or disgraceful conduct or conduct which when regard is had to your profession is improper or disgraceful in that

(i) before rendering professional services to one E.J.G. in or about the year 1953, you failed to inform him of the fee which you intended to charge namely, £400, which fee exceeds that usually charged for the services which you rendered, the circumstances being such that it was not possible for you to do so;

(ii) in charging the said E.J.G. in or about the year 1953 the sum of £400 in respect of the services rendered, you recovered or attempted to recover excessive fees;

(iii) you unjustifiably cast reflection by word or implication upon the professional reputation and skill of your fellow practitioners, namely, all practitioners on the medical register, by communicating with the said E.J.G. in a letter dated 5 August 1953, of which the following is a copy.

'Please excuse the delay in answering your letter. I am delighted to hear that your leg is much more comfortable; you will soon have the pleasure of feeling that you have a brand new leg.

'Your recovery from the two operations that you have undergone has been so smooth that you are possibly unaware of the fact that a tremendous piece of work has been done for you. The first operation lasted 2½ hours, and the second 3½ hours. The second operation was of such a magnitude that had I not done it you would have been forced to visit Boston, U.S.A., for its execution or become increasingly crippled. Now I am sure that a man of your talents and interest in sport would not simply sit down and await increasing crippledom. You would undoubtedly, since you are young and otherwise vigorous, have made plans to have the operation done in the U.S.A., an act that would have involved you in the expenditure of £5,000, for you would have been away from South Africa for many months, your wife with you, and as you know medical and surgical fees in the U.S.A. are 5 to 10 times our fees in South Africa.

'I have done a perfect piece of work for you, a tremendous undertaking that has spared you a visit to the U.S.A. and has therefore saved you considerable expense and time away from your business. You are well off financially, you occupy an honoured place in Society, and on all sides I hear of your successful reputation in the world of business.

'You would under the circumstances surely not begrudge me a fee of £400 for an *exclusive* operation? The benefit derived from this operation will transform your life and restore to you your former health and happiness! Is not this prospect one providing the greatest pleasure? I look forward to seeing you on Sunday 9 August, at the Florence Nightingale Nursing Home, when I shall renew your leg cast.

With best wishes.'

"Alternatively to (iii): (iv) in communication with the said E.J.G. in a letter dated 5 August 1953, a copy whereof appears in paragraph (iii) above, you acted improperly or disgracefully or in a way unbecoming to a medical practitioner.'

The Council resolved that Dr. G. be found guilty of disgraceful conduct on count (ii) and be found guilty of conduct which when regard is had to his profession is improper on count (iii); he was found not guilty on count (i); having been found guilty on count (iii), the alternative charge (i.e. (iv)) fell away.

The Council resolved that Dr. N.G. be suspended from practice as a medical practitioner for a period of 3 months as from 15 October 1955.

PASSING EVENTS : IN DIE VERBYGAAN

Union Department of Health Bulletin. Report for the 7 days ended 6 October 1955.

Plague, Smallpox, Typhus Fever: Nil.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Dacca (Pakistan).

Smallpox in Kabul (Afghanistan); Moulmein, Rangoon (Burma); Phnom-Penh (Cambodia); Kozhikode, Madras, Tellicherry (India); Dacca, Lahore (Pakistan); Saigon-Cholon (Viêt-Nam).

Typhus Fever: Nil.

An Obstetric week end Refresher Course conducted during September by the Department of Gynaecology of Natal University was attended by 130 general practitioners from central and remote parts of Natal and Zululand. This first clinical post-graduate venture is the precursor of regular refresher courses for general practitioners conducted by the entire Medical School, which will commence in March or July 1956. The Principal, Dr. E. G. Malherbe, opened the proceedings and welcomed the guests on behalf of the University of Natal. He was followed by the visiting lecturer, Professor E. C. Crichton of Cape Town, who gave an address on

Induction of Labour. Further lectures were given by Mr. D. F. Standing, Mr. A. L. Gilbey, Dr. G. Were and Professor Derk Crichton.

* * *

The East Rand Branch of the Medical Association of South Africa will hold a Dinner-Dance at the Alexandra Hotel, Germiston, on Friday, 18 November 1955, at 8 p.m.

* * *

The Kalinga Prize, offered annually by the United Nations Educational, Scientific and Cultural Organization, has been awarded for 1955 to Dr. August Pi Sûner (Spain), Director of the Institute of Experimental Medicine of the University of Caraças, Venezuela. The object of the £1,000 prize is to offer recognition to leading interpreters of science and also to strengthen links between India and scientists of all nations.

* * *

The 10th International Congress of the International College of Surgeons will be held in Chicago, Illinois, September 9-13, 1956, in conjunction with the 21st annual congress of the United States and Canadian sections. The Buenos Aires meeting, previously announced, has been cancelled.

* * *

Owing to unforeseen circumstances the lecture which Professor G. A. Elliott was to have delivered under the auspices of the General Practitioners' Section of the Southern Transvaal Branch of the Association at Medical House, Esselen Street, Hospital Hill, Johannesburg on 27 October had to be postponed until some time next year. Professor Elliott's subject will be *Psychological Medicine in General Practice.*

* * *

International Science Foundation USA: Scholarship and Fellowship Programme for Overseas Scientists. The South African Council for Scientific and Industrial Research has received information concerning provision made by the California Academy of Sciences, Golden Gate Park, San Francisco, for the assistance of overseas scientists desiring to do graduate work in US universities. This committee will provide information concerning the US universities offering the best academic opportunities in the areas of specialization of interest to each applicant. Whenever possible, the committee will assist the applicant in obtaining a fellowship or scholarship from the university of his choice and will also process requests for subsistence grants from corporate or other sources.

* * *

The 9th General Assembly of the World Medical Association which convened in Vienna, Austria, September 20-26, 1955, was the best attended, most enthusiastic and interesting annual meeting the association has ever held. Prof. Dr. A. Spinelli of Italy presided at the opening session and inaugurated Dr. Karl Niederberger of Austria as President for 1955-1956. The venue of the 10th General Assembly was set for Havana, Cuba, to be convened October 9-15, 1956.

GERMAN SCHOLARSHIPS FOR SOUTH AFRICAN STUDENTS

The Department of Education, Arts and Science has been advised by the German Academic Exchange Service (Deutsche Akademische Austauschdienst) that bursaries are available to South African students who wish to study at a university or a high school in the Federal Republic of Germany or in Western Berlin.

The following conditions apply to these bursaries:

(1) The bursaries are for the period running from 1 November 1956 to 31 October 1957. They amount to 3,000 German marks each and will be paid out in advance in monthly instalments.

The scholarships also include an amount equal to the cost of travelling from the German border to the place of study and back to the border.

The Deutsche Akademische Austauschdienst will furthermore urge the universities concerned to exempt bursars from payment of class fees as far as possible.

(2) South African Students of European descent who have completed two years successful study at a South African university and who have a good working knowledge of German, may apply for the scholarships.

(3) Application forms which are obtainable from the Department of Education, Arts and Science, Van der Stel Buildings, Pretoria, must be duly completed and accompanied by supporting papers and must reach the Department not later than 3 December 1955.

BOOK REVIEWS : BOEKRESENSIES

PERIPHERAL VASCULAR DISEASES

Peripheral Vascular Diseases. By Edgar V. Allen, B.S., M.A., M.D., M.S. in Medicine, F.A.C.P., Nelson W. Barker, B.A., M.D., M.S. in Medicine, F.A.C.P., Edgar A. Hines, Jr., B.S., M.A., M.D., M.S. in Medicine, F.A.C.P., with Associates in the Mayo Clinic and Mayo Foundation. Second Edition. Pp. 825+xiii with 316 illustrations. Philadelphia & London: W. B. Saunders Company. 1955.

Contents: 1. Definition of Terms. 2. An Approach to the Diagnosis of Vascular Diseases of the Extremities. 3. Special Methods of Investigation. 4. Raynaud's Phenomenon and Allied Vasospastic Conditions. 5. Scleroderma. 6. Diseases of the Vascular System Which Primarily Result from Environmental Temperature. 7. Erythermalgia. 8. The Scalenus Anticus Syndrome with and without Cervical Rib. 9. Sudden Occlusion of the Arteries. 10. The Degenerative Arteriopathies: Arteriosclerosis and Atherosclerosis. 11. Arteriosclerosis Obliterans. 12. Thromboangiitis Obliterans. 13. Coarctation of the Aorta. 14. Periarteritis Nodosa and Temporal Arteritis. 15. Other Types of Local and Disseminated Arteritis. 16. Hypertensive Ischemic Ulcers of the Leg. 17. Aneurysms. 18. Arteriovenous Fistulas. 19. Tumors of Blood and Lymph Vessels. 20. Purpura. 21. Diseases of the Veins: General Considerations and Various Diseases Exclusive of Thrombophlebitis, Arteriovenous Fistula and Varicose Veins. 22. Thrombophlebitis. 23. Varicose Veins. 24. Chronic Venous Insufficiency. 25. Lymphedema. 26. Medical Treatment. 27. Surgical Treatment. 28. Surgical Procedures on the Sympathetic Nervous System for Peripheral Vascular Disease. 29. Injection of Local Anaesthetic Agents and Alcohol into Ganglia of Sympathetic Nerves to the Extremities. 30. Amputations for Peripheral Vascular Disease. 31. Nonvascular Operations for Intermittent Claudication. 32. The Ingrown Toenail and Corns. 33. Embolectomy, Arteriectomy and Thromboendarterectomy. 34. Surgical Treatment of Coarctation of the Aorta. 35. Surgical Treatment of Aneurysms of the Aorta. 36. Surgical Treatment of Thrombotic Occlusion of the Terminal Portion of the Aorta. 37. Surgical Treatment of Peripheral Arterial Aneurysms. 38. Surgical Treatment of Arteriovenous Fistulas of the Peripheral Vessels. 39. Vascular Injuries in the Extremities. 40. Surgical Treatment of Varicose Veins. 41. Sclerosing Therapy. 42. Skin Grafting for Stasis Ulcers. 43. Surgical Treatment of Lymphedema. Index.

What do we mean by Peripheral Vascular Disease? The premise in the foreward to this book that the term should include disease of all vessels distal to the heart is at least reassuring in that it defines the limits of this hitherto rather vague subject—also a little alarming when one comes to appreciate the extent of the field which these boundaries seek to contain.

So much new work has been done in this previously neglected branch of medicine since the first edition was published in 1946, that the authors must have been hard put to it to decide how much of the old and trite to discard to make room for the new and interesting. The changes that have been made are mostly in favour of the clinical and practical at the expense of the academic and theoretical. Thus we find that, in the chapter on Special Methods of Investigation, such subjects as plethysomography, capillaroscopy and others received but a passing mention, while those that are of practical use to the clinician, such as angeography, are elaborated and emphasized.

This feature is even more apparent in the section on treatment. The chapter on Medical Treatment has been considerably (though we feel not adequately) curtailed; whilst that on Surgical Treatment has been much expanded—from 64 pages in the first edition to 137 in this, the second. May it be a happy augury to sufferers from peripheral vascular disease that, whereas the former begins gloomily with amputations, the latter opens on a more cheerful note—the potentialities of surgery on the sympathetic and arterial systems.

This new section gives a comprehensive account of modern operations on the sympathetic ganglia—concise yet adequately technical and admirably illustrated. Details of such simple yet essential procedures as anaesthetic block of the ganglia have not been neglected. Other surgical procedures such as thrombendarterectomy, resection of aneurysms and coarctation of the aorta are dealt with in an adequate yet restrained manner.

A general impression gained from this edition is that certain conditions are in the process of being clarified and brought into clearer focus. The various vasospastic states, for instance, once vagrant and indeterminate, seem to be settling down happily in the Reynaud *ménage*. The inclusion of Purpura would appear to be out of focus; it is difficult to see why the authors should be concerned with the contents rather than the containers.

This book is not the expression of a single individual, but a collection of essays by a group of men, each one an expert in his own sphere. The general format is refreshing, the somewhat desiccated atmosphere of the average text-book being dispelled by the presence as an introduction to each chapter of a portrait and biographical note of some pioneer.

On the whole this is an admirable book, comprehensive, clear and concise, and it should find a place on the book-shelf of the general practitioner as well as the physician and surgeon.

M.A.L.

OLD AGE: A SYMPOSIUM

Old Age in the Modern World. Report of the Third Congress of the International Association of Gerontology, London, 1954. Pp. 647+vii with tables. 35s. Edinburgh & London: E. & S. Livingstone Ltd. 1955.

Contents: 1. Age and Society. 2. National Programmes. 3. Integration of Plans for the Care of the Aged in Health and Infirmity in the United Kingdom. 4. Nutritional Aspects of Ageing. 5. Tissue Changes. 6. Cancer. 7. Endocrinology. 8. Experimental Studies of Changes in Performance with Age. 9. Employment of Older Workers. 10. Surveys. 11. Neuropsychiatry. 12. Cardiovascular Diseases. 13. Clinical Problems. 14. Residential Care. 15. Community Activities. 16. Education for Later Maturity in the U.S.A. 17. General Aspects of Ageing. Indices.

The publication of this work on the problems presented by an ageing population will be heralded without a doubt as a most significant advance in our knowledge of the subject. There is hardly an aspect of the problem of old age which has not been exhaustively dealt with by the various contributors to the volume. The keynote of the theme was struck by Dr. J. H. Sheldon, of Wolverhampton, in his Presidential Address entitled *The Social Philosophy of Old Age*. 'The basic problems of old age', he states, 'are common to all countries, and everywhere they are commanding the attention of administrators, economists, social scientists, and physicians'.

These problems, indeed, can no longer be evaded, for the reason that their gravity is increasing with the march of the years. I consider that the average medical reader, bent on a holistic approach to the study of biosocial phenomena, will be intrigued as much by the sociodemographic as by the medical aspects of the problem. For several decades now there has been both an absolute and a relative increase in the number of aged persons in the population, i.e. of men and women 65 years and over. This increase has been due to the simultaneous operation of two factors, namely, the decline of the birth rate (from about 1870 onwards), and the steady decline in the death rate. An idea of the magnitude which the problem will assume in Great Britain in the future can be obtained from the estimate of the Royal Commission on Population, namely that in 1977 there will be a 46% increase of old persons over the 1947 figures, i.e. an increase from 5 million to 7·3 million. But this increase in the number of aged persons, in so far as it will not be accompanied by a corresponding increase in the number of persons in the younger age-groups, means that there will be progressively fewer persons of working age available for the burden of supporting the increasing number of aged persons.

This, however, may appear to be only the economic aspect of the problem, but then there are other inter-connected problems which an ageing population brings in its train, such as a sharp increase in the incidence of neoplasia, of degenerative vascular states, of physical disability, and of mental disorder.

It is apparent that the problem presented by an ageing population is multiplex, and calls for a simultaneous approach at the medical, medico-social, and medico-sociological level, as has been advocated by the reviewer in his lectures on Social Medicine at the University of Witwatersrand. A similar approach is being followed by at least two of the contributors to the Symposium, namely, Dr. Anitra Karstan, of Helsinki, and Professor E. W. Burgess, of Chicago. Perhaps this approach will one day bring it about that Crabbed Age and Youth can live together, though youth be full of pleasance, and age full of care.

Medical practitioners everywhere will be under a deep debt of gratitude to the British Organizing Committee of the Third Congress of the International Association of Gerontology for their sponsorship of this most scholarly and encyclopaedic work.

L.F.F.

NURSING PROCEDURES

Illustrated Practical Nursing Procedures for Hospital Assistants. By Josephine Scott, S.R.N., S.C.M., S.R.M.N. Pp. 118+ix with 64 illustrations. 15s. London: William Heinemann Medical Books Ltd. 1955.

Contents: 1. Hospital Etiquette. 2. General War Work. 3. Admission of new patients. 4. Bed-making. 5. Positions used in nursing. 6. Care of the patient. 7. Temperature, pulse and respiration. 8. Bathing in the bathroom. 9. Enemata. 10. Local applications. 11. Lavage and Catheterization. 12. Artificial feeding. 13. Aspiration and artificial pneumothorax. 14. Syringing of the ears. 15. Wounds. 16. Fractures. 17. Preparing the patients for operation. 18. Sterilization and Asepsis. 19. Fluid Balance. 20. Administration of Medicines and Drugs. 21. Collecting material for laboratory examination. 22. Operations and general set of instruments. 23. Care of the dead. Index.

This is an extremely well prepared book. The script is well set out and easy to follow. Miss Scott has used appropriate illustrations freely, and this is certainly a great help in a book on *practical procedures*. One is, perhaps, somewhat surprised at the very advanced techniques to be carried out by hospital *assistants*. But this book is presumably meant for trainees of the African Medical Training School at Lusaka and, although the subject matter is essentially '*nursing procedures*', the author probably had in mind the type of hospital assistant who would appreciate this book and would be a person who had been very carefully selected and trained—a stage between the State Registered Nurse and the Nursing Assistant.

This book does not attempt to be a complete text-book, but its 114 pages are full of valuable and clearly set-out information.

THE DIABETIC LIFE

The Diabetic Life. Its Control by Diet and Insulin. A Concise Practical Manual for Practitioners and Patients. By R. D. Lawrence, M.A., M.D., F.R.C.P. (London). Fifteenth Edition. Pp. 228+xii with 19 illustrations. 12s. 6d. London: J. & A. Churchill Ltd. 1955.

Contents: 1. A Comparison of Normal and Diabetic Metabolism. 2. The Causation and Pathology of Diabetes and the Discovery of Insulin. 3. The Symptoms of Diabetes. 4. Diagnosis and Blood Sugar Tests. 5. Principles of Treatment by Diet and Insulin: Who Needs Insulin? 6. Diet Schemes for Treatment: The Line-Ration Scheme; Treatment of Mild Cases by Weighed Diet; Treatment by Simple Unweighed Diet; Reducing Diet. 7. Principles of Insulin Treatment: The Qualities and Action of Different Insulin Preparations; The New Insulin Zinc Suspensions. 8. Actual Treatment with Insulin: Lente Insulin; Globin, N.P.H., Semi-Lente; Protamine Zinc Insulin; Soluble Insulin; Emergency Treatment of Ketosis; Exercise and Diet Adjustments; Clinical Response and Progress; The Choice and Type of Insulin. 9. Further Problems in Diabetic Management: Future Progress and Prognosis; A Cure and 'Cures'; Different Ideals and Methods of Treatment; The Minimum Essentials of Insulin Treatment; Ideal Weight and Weight Control; The Omission of Insulin; The Relation and Relative Value of Urine and Blood-Sugar Tests; Insulin Sensitivity and Resistance. 10. Insulin Injections: Difficulties in Injection; Local Reactions. 11. Hypoglycaemia and its Treatment; Endogenous Hypoglycaemia. 12. Treatment of Coma and Precomatose Conditions. 13. Treatment of Children. 14. Control of Variations and Complications in Treatment; Treatment of Complicating Illnesses;

Complications of Diabetes—Eye Complications and Retinopathy; Neuropathy; Mental Balance; Vascular and Renal Complications; Pregnancy; Heredity and Marriage. 15. Surgical Complications; Choice of Anaesthetic; Gangrene; Types of Foot Lesions; Carbuncles. 16. Miscellaneous: Oedema and Dropsy; Pigmentation of Skin; Other Skin Conditions; Pancreatic Disease; Lipaemia and Hypercholesterolaemia; Vitamins; Contra-Indications to Insulin. 17. The Choice and Management of Diet. 18. Diabetic Recipes. 19. The Essentials of a Diabetic Education; Rules of General Health; Care of the Feet; Treatment of Minor Injuries; Precautions for Motor Driving. 20. The Five-Gram Diet Scheme and Food Tables; Other Miscellaneous Information. Appendix. Index.

'The Diabetic Life' hardly needs a review; it is too well-known. As Lawrence remarks, it is a book for patient and doctor, and both its originality and its danger spring from this. It is good for many diabetics to understand their disability, but for many others it is far preferable not to read too much about it. It is not always the most intelligent to whom this book should be given to read, as those who deal with diabetics find out to their cost!

Now as regards doctor-readers: One gets the impression that very little revision has gone into this edition, as indicated by the odd and quite inadequate footnotes (e.g. p. 140 on complicating pneumonia; 'give antibiotics at once, not the sulpha drugs'). Obsolescent Z.P.I. is still discussed in detail, while the brief remarks on the new insulins are somewhat confusing. Quite remarkable caution is shown in the use of insulin in coma, and in preparation of a patient for operation. Serum ketone examination is not mentioned.

There is, of course, the same good practical advice as always, and there is no doubt that all physicians and general practitioners should be acquainted with this book.

P.J.

SKIN DISEASES IN GENERAL PRACTICE

General Practice Series: Common Skin Diseases. By A. C. Roxburgh, M.A., M.D., B.Ch. (Cantab.), F.R.C.P. (London). Tenth edition. Pp. 515 + xxi, with 215 illustrations and 8 plates. £1 10s. 0d. London: H. K. Lewis & Co. Ltd. 1955.

Contents: 1. General Etiology and Pathology. 2. Signs Symptoms and General Diagnosis. 3. General Treatment. 4. Congenital Affections of the Skin. 5. Skin Affections due to Physical Causes. 6. Dermatitis due to Chemical Causes. 7. Drug Eruptions. 8. Diseases due to Animal Parasites. 9. Diseases due to Vegetable Parasites. 10. Diseases due to Filterable Viruses. 11. Syphilis. 12. Diseases due to Obstruction of Vessels. 13. Neurodermatoses. 14. Toxic Eruptions. 15. Eczema, Besnier's Prurigo, Pompholyx, Acrodermatitis Perstans. 16. Psoriasis and Pityriasis Rosea. 17. Lichen Planus. 18. Diseases of Hair and Nails. 19. Diseases of Sebaceous and Sweat Glands. 20. Tumours. 21. Atrophy and Sclerosis. 22. Vesicular and Bullous Eruptions. 23. The Erythrodermias. 24. Avitaminoses. Index.

Ten editions and several reprintings in the last 20 years testify to the popularity of this book with students and practitioners. This latest edition, the last to be put through the press by the late Dr. Roxburgh, is basically unchanged but contains much new material. Good clinical descriptions and ample illustrations have always been the basis for Roxburgh's popularity.

Not everyone will agree with the method of classification, and some diseases, such as those of the reticulo-endothelial system, are poorly served. Histopathology is now recognized as a basis and essential part of dermatology but it still receives far too little attention here.

In spite of these shortcomings there is no doubt that this is stil. one of the best short British books on the common skin diseases.

J.M

CORRESPONDENCE : BRIEWERUBRIEK

PORPHYRIA IN THE BANTU RACES ON THE WITWATERSRAND

To the Editor: My attention has been drawn to an error in connection with Fig. 5 in my paper under the above title in the issue of the *Journal* for 20 August 1955.

This illustration was included as a result of a misunderstanding and should not have appeared as it is not relevant to the subject of the paper.

H. D. Barnes

South African Institute for Medical Research
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Johannesburg
11 October 1955

SPECIALIST AND CONSULTANT REGISTER

To the Editor: Several weeks ago the local Press blazoned in front page headlines the impending replacement of the Specialist Register by a Consultant Register and gave a rehash of a leading article in a contemporary medical journal.

Whether this article was intended to influence the Medical Council is a moot point, but the resultant vote taken at the Medical Council was most disturbing and distressing to a large section of the medical profession.

As a junior specialist who has just succeeded in getting on the Specialist Register after 8 years of intensive postgraduate work overseas and in hospitals, I am most perturbed at the mere possibility of such a change. I certainly would not have given up general practice and sacrificed 8 valuable years had I contemplated even the remote possibility of such a change.

Such a reversal would be calamitous to the great majority of specialists practising, and to the many hundreds who are studying to become specialists. Are they all to be sacrificed in order to satisfy the whims and fancies of the general Practitioners Group?

The Medical Association has insisted as priority No. 1 on the free choice of doctor by each patient, and has for years waged and is still waging a battle against Medical Benefit Societies with closed panels. Such a Consultant Register surely deprives the patient of a free choice of doctor.

I will not at this stage enlarge on the question of diversion of patients and dichotomy—two very important aspects of practice if a Consultant Register ever does replace a Specialist Register—and please do not let the protagonists of the Consultant Register shudder at the mention of these two 'syndromes'.

The implications and repercussions of a change from a Specialist to a Consultant Register will rock the profession to its very foundations—and incidentally will not improve the status or the income of the general practitioner.

Junior Specialist

Johannesburg
12 October 1955

THE SOUTH AFRICAN POLIO VACCINE

To the Editor: I have followed the articles and correspondence in the *Journal* on poliomyelitis vaccination with the interest the question deserves and demands from one as a medical man and as a parent. The constitution of the committee advising the Minister on our South African vaccine is such that it would be very difficult to add weight to the value of its advice.

Maybe a communication to the profession in this country like the very excellent one by Turner[1] might more happily have been made a bit earlier. But now that we are aware of the facts as they affect us in this country, with the possible snags and the imperfections there for all to see, each of us has his mind to make up.

Is Dr. Maurice Shapiro[2] not perhaps reading the looking glass book a bit with Alice?

> 'Beware the Jabberwock, my son!
> The jaws that bite, the claws that catch!
> Beware the Jubjub bird, and shun
> The frumious Bandersnatch!'

In his scientific zeal, the vaccine in the shape of the Jabberwock, the simian kidney tissue taking feathers upon itself, and the Bandersnatch penicillin, make a somewhat horrific trio. But the practice of medicine involves taking the human road as well, and I find it difficult to regard those working at the Polio Research Foundation, and the men on the committee advising on the safety of the vaccine, as 'slithy toves', who 'gyre and gimble in the wabe'.

With apologies to Lewis Carroll.

K. Sartorius

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7 October 1955

1. Turner, R. (1955): S. Afr. Med. J., **29**, 833.
2. Shapiro, M. (1955): *Ibid.*, **29**, 947.